Periimplantitis- A review

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ABSTRACT
This review article concentrates the light about aetiology and treatment of the periimplantitis. (J Bagh Coll Dentistry 2015; 27(2):101-104).

INTRODUCTION
The goal of modern dentistry is to restore the patient to normal contour, function, comfort, esthetics, speech, and health, regardless of the atrophy, disease or injury of stomatognathic system. Teeth are integral part of the stomatognathic system. The primary function of teeth is to prepare food for swallowing as well as to initiate and facilitate digestion. Teeth are also necessary for the articulation of speech and proper looks. Implant-based dental rehabilitation techniques has come to offer highly predictable results, hence it has become one more element to be included in the wide range of therapeutic alternatives for totally or partially edentulous patients, albeit some complications have been described in relation with this type of treatment; of these complications, the progressive loss of alveolar bone surrounding the implant is perhaps the most salient. The name periimplant disease refers to the pathological inflammatory changes that take place in the tissue surrounding a loadbearing implant (1) for some authors it is the most common complication in oro-facial implantology (2).

Two entities are described within the concept of periimplant disease: - Mucositis: a clinical manifestation characterized by the appearance of inflammatory changes restricted to the periimplant mucosa. If treated properly, it is a reversible process (3). Periimplantitis: a clinical manifestation where clinically and radiologically evident loss of the bony support for the implant occurs, together with an inflammatory reaction of the periimplant mucosa (4).

Etiopathogeny of periimplantitis
1- Periimplant tissue morphology: - Healthy periimplant tissue plays an important role as a biological barrier to some of the agents that cause periimplant disease. The epithelium and the interface between the supraveolar connective tissue and the titanium surface of an implant differ from the interface of the dental-gingival unit. Like the connective tissue attachment, the epithelium presents a hemidesmosomal attachment to the implant surface; the difference lies in the fact that the epithelial fibers are predominantly longitudinal to the surface of the implant and not perpendicular, as in the case of a natural tooth. In the most coronal region, they are circumferential, in addition to presenting a low degree of vascularization and a higher collagen fiber to fibroblast ratio in comparison to the tooth (a ratio of 4 in a tooth to 109 in the implant) (5).

2- Implant structure: - The design of the implant is an important factor in the onset and development of periimplantitis. Poor alignment of the components that comprise an implant prosthesis system may foster the retention of bacterial plaque, as well as enabling microorganisms to pass inside the transepithelial abutment.

3- Microbial infection: - Another cause of periimplantitis, as previously mentioned, is the bacterial colonization of the periimplant pocket. The association between different microorganisms and destructive periodontal or periimplant disease is governed by the same biological parameters. The microorganisms most commonly related to the failure of an implant are the Gram negative anaerobes, like Prevotella intermedia, Porphyromonas gingivalis, Actinobacillus actinomycetemcomitans, Bacteroides forsythus, Treponema denticola, Prevotellanaigrans, Peptostreptococcus micros and Fusobacterium nucleatum (6).

4- Excessive mechanical stress: - Another factor that intervenes in periimplantitis aetiopathogeny is excessive
mechanical stress. The process begins with the appearance of microfractures of the bone around an osseointegrated implant, as a result of being subjected to axial or lateral stresses that are excessive for its load-bearing capacity. On occasions, these forces cause a prosthetic component (resin, ceramic or the transepithelial abutment screw) or the implant itself to fracture, without any loss of bone height or osseointegration whatsoever.

**Diagnoses, Prevalence, and Incidence**

From a clinical standpoint, signs that determine the presence of peri-implant mucositis include bleeding on probing and/or suppuration, which are usually associated with probing depths +4 mm and no evidence of radiographic loss of bone beyond bone remodeling. Outcomes from reports (7,8) assessing the prevalence of peri-implant diseases revealed that peri-implant mucositis was present in 48% of implants followed from 9 to 14 years affected with this problem.

Since peri-implant mucositis is reversible with early intervention and removal of etiology, it is quite possible that its prevalence could be under reported. However, when these same parameters are present with any degree of detectable bone loss following the initial bone remodeling after implant placement, a diagnosis of peri-implantitis is made. Peri-implantitis can be diagnosed early or once clear clinical evidence has developed. The most common signs and symptoms are:

- Color changes in keratinized gum tissue or in the oral mucosa.
- Bleeding on probing.
- Increased probing depth of periimplant pockets.
- Suppuration.
- Periimplant radiotransparency.
- Progressive loss of bone height around the implant.

The absence of bleeding on probing is indicative of good health. Probing depth depends on the force applied, so that when equal amounts of force are exerted, the depth reached by the probe is greater in periimplantitis than in the case of a natural tooth. It is recommended the use of probes calibrated to a force of 0.25 n (25 g) to avoid test errors. At any rate, a pocket larger than 5 mm is deemed to have a greater likelihood of being contaminated. On x-ray, the problem can be detected once 30% of the bone mass has been lost; hence this is not an optimal method for early diagnosis of periimplantitis.

Distinct differences in the incidence and prevalence of peri-implantitis have been reported by a number of authors. Most recently, a publication discussed this problem and noted that a literature search of 12 studies in which bleeding on probing and/or purulence were detected with concomitant radiographic bone loss, revealed eight different thresholds of radiographic bone loss used as a disease criteria. This has led to a variation in the reported prevalence of periimplantitis around implants. For example, one study found the prevalence to be 6.61% over a 9 to 14 year period (9), another 23% during 10 years of observation (9), and a third reported a prevalence of 36.6% with a mean of 8.4 years of loading (10).

The problem with applying differing thresholds for probing depth and radiographic bone loss to define peri-implantitis has been discussed in explaining the variance in reporting the prevalence of peri-implantitis. In one study, the prevalence varied from approximately 11% to 47% of subjects depending on the threshold used (10). Although it requires evidence based studies for validation, a peri-implant disease classification has been proposed to aid in explaining disease severity and threshold.

**Risk Factors**

A number of risk factors have been identified that may lead to the establishment and progression of peri-implant mucositis and peri-implantitis (11). The following are some of those factors:

1- Previous Periodontal Disease: - Systematic reviews (12-15) have indicated that although the implant survival rate may not be affected by the periodontal history, peri-implantitis was a more frequent finding in patients with a history of periodontitis.

2- Poor Plaque Control/Inability to Clean: - Implant prosthesis design can obviate the patient’s ability to mechanically clean the site with brushes, interdental brush, and floss. This can be related to implant positioning and meeting patient expectations for esthetics, phonetics, and function. Moreover, prosthesis design can also preclude clinical evaluation with probing and adequate home-care procedures (16).

3- Residual Cement: - A growing area of concern has been the incomplete removal of cement left in the subgingival space around dental implants (17).
4- Smoking: - Four systematic reviews have concluded that there is an increased risk for peri-implantitis in smokers, with odds ratios ranging from 3.6 to 4.6 (12).

5- Genetic Factors: - Genetic variations have been cited as a risk factor for peri-implantitis. However, the association between IL-1 gene polymorphism and peri-implantitis remains to be determined since conflicting results exist.

6- Diabetes: - The evidence regarding the association between diabetes and peri-implantitis is limited because of the small number of studies.

7- Occlusal Overload: - One of the difficulties in conducting clinical studies on this topic rests on the definition of occlusal overload. Differences in the magnitude, duration, direction, and frequency of the applied occlusal load and the tolerance threshold of the host are the underlying reasons of the observed conflicting reports. Possible mechanisms of why occlusal overload can lead to peri-implantitis are conceivable. Implants are considered less tolerable to non-axial occlusal load compared to teeth because of a lack of a periodontal ligament.

8- Potential Emerging Risk Factors: - Research endeavors continue to explore some additional areas that may impact the development and pathogenesis of peri-implantitis. These include rheumatoid arthritis with concomitant connective tissue disease, increased time of loading, and alcohol consumption. Further study will determine the appropriateness of their inclusion.

The Goals of Treatment of Periimplantitis

1- Regeneration of bone structures; complete elimination of inflammatory processes in the peri-implant tissues.

2- Reduction in the duration of the treatment.

3- Creation of aseptic conditions around the implant.

4- Securing the reliability of the implanted artificial supports.

The criteria of the treatment

• Early, the chances of success are best.
• Supported by procedures designed to lead to the improvement towards healing.
  • Simple removal of local factors is not sufficient.
• Supported and complemented by surgical and biostimulation procedures.
• Complex procedures – antibiotics and anti-inflammatory drugs.
• Surgical procedures.
• Restoration of teeth and arch morphology.
• Occlusion balancing.
• The more diversified the disease, the more it shows an advanced degree in evolution.
• Designed for each individual, is the main condition of success, to improve the condition, to obtain healing.
• Treatment of periodontal disease must take into account the general condition of the patient as periodontal treatment can be both local and general.

Nonsurgical Treatment of Peri-Implantitis

A- Mechanical treatments

Karring et al. compared the results compared the treatment results obtained with the Vector® ultrasound system and with carbon fiber curettes (18). After 6 months of follow-up, no significant differences were found between the two techniques, and neither proved sufficient to treat peri-implantitis. These authors evaluated 31 patients, comparing ultrasound (Vector® system) and mechanical treatment with curettes. After 6 months, both study groups showed improvement in plaque index and bleeding, though without improvement in terms of pocket depth. There were no significant differences between the groups, and the changes recorded were of no clinical relevance. In relation to bacterial load, there were no differences in the change in bacterial composition in the two groups after treatment.

B- Mechanical treatments associated to antibiotics

The recommended antibiotic treatments are amoxicillin, amoxicillin plus clavulanic acid, amoxicillin plus metronidazole, or erythromycin plus tetracycline, with a duration of 7-10 days. The selected articles examined treatment with minocycline microspheres, the use of doxycycline, and the administration of metronidazole.

Surgical Treatment of peri-implantitis

A- Resection techniques

Resection techniques are used when there are moderate (< 3 mm) horizontal suprabony defects or vestibular dehiscences in a non-aesthetically compromised region. These procedures include ostectomy or osteoplasty, with the raising of an apical repositioning flap and implantoplasty.
B- Regenerative surgery

Regenerative surgery is used when the implant is decisive for prosthetic preservation, or when aesthetic considerations are involved. Regenerative treatment requires prior decontamination of the implant surface. Most studies use the concept of guided bone surgery, which includes the placement of a membrane after grafting. Many bone substitutes are available, though very few randomized trials have compared them in the context of the treatment of peri-implantitis.

Conclusions

Most of the factors that lead to implant failure can be controlled by the dentist by means of proper treatment planning prior to implant surgery. The number, diameter and location of the implants depending upon patient bone type and the type of prosthesis to be inserted, are all factors that are clearly within our control. Patients undergoing chronic corticoid therapy, poorly controlled diabetics, smokers, those who present active periodontal disease and individuals with serious systemic pathology or predisposing genetic factors should be considered high-risk cases. Prognosis of the affected implant will be contingent upon early detection and treatment of mucositis and periimplantitis.

REFERENCES